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NEWS 2	Apr 08	"Ask CAS" for self-help around the clock
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NEWS 4	Apr 09	ZDB will be removed from STN
NEWS 5	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 6	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 7	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 8	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 9	Jun 03	New e-mail delivery for search results now available
NEWS 10	Jun 10	MEDLINE Reload
NEWS 11	Jun 10	PCTFULL has been reloaded
NEWS 12	Jul 02	FOREGE no longer contains STANDARDS file segment
NEWS 13	Jul 22	USAN to be reloaded July 28, 2002; saved answer sets no longer valid
NEWS 14	Jul 29	Enhanced polymer searching in REGISTRY
NEWS 15	Jul 30	NETFIRST to be removed from STN
NEWS 16	Aug 08	CANCERLIT reload
NEWS 17	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS 18	Aug 08	NTIS has been reloaded and enhanced
NEWS 19	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
NEWS 20	Aug 19	IFIPAT, IFICDB, and IFIUDB have been reloaded
NEWS 21	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 22	Aug 26	Sequence searching in REGISTRY enhanced
NEWS 23	Sep 03	JAPIO has been reloaded and enhanced
NEWS 24	Sep 16	Experimental properties added to the REGISTRY file
NEWS 25	Sep 16	Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 26	Sep 16	CA Section Thesaurus available in CAPLUS and CA
NEWS 27	Oct 01	CASREACT Enriched with Reactions from 1907 to 1985
NEWS 28	Oct 21	EVENTLINE has been reloaded
NEWS 29	Oct 24	BEILSTEIN adds new search fields
NEWS 30	Oct 24	Nutraceuticals International (NUTRACEUT) now available on STN
NEWS 31	Oct 25	MEDLINE SDI run of October 8, 2002
NEWS 32	Nov 18	DKILIT has been renamed APOLLIT
NEWS 33	Nov 25	More calculated properties added to REGISTRY
NEWS 34	Dec 02	TIBKAT will be removed from STN
NEWS 35	Dec 04	CSA files on STN

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FILE 'HOME' ENTERED AT 10:09:24 ON 10 DEC 2002

=> file biosis
COST IN U.S. DOLLARS

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FULL ESTIMATED COST	0.21	0.21

FILE 'BIOSIS' ENTERED AT 10:09:37 ON 10 DEC 2002
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=> s alpha(W)sub(W)1
        568577 ALPHA
           299 ALPHAS
        568687 ALPHA
                  (ALPHA OR ALPHAS)
         53200 SUB
            40 SUBS
         53237 SUB
                  (SUB OR SUBS)
       2809819 1
L1           1 ALPHA(W)SUB(W)1

=> s alpha(W)sup(W)1
        568577 ALPHA
           299 ALPHAS
        568687 ALPHA
                  (ALPHA OR ALPHAS)
           946 SUP
            18 SUPS
           960 SUP
                  (SUP OR SUPS)
       2809819 1
L2           0 ALPHA(W)SUP(W)1

=>  s alpha(W)1
        568577 ALPHA
           299 ALPHAS
        568687 ALPHA
                  (ALPHA OR ALPHAS)
       2809819 1
L3       47735 ALPHA(W)1

=>  s alpha1
L4        9573 ALPHA1
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=> s l1 or l2 or l3 or l4
L5        52058 L1 OR L2 OR L3 OR L4

=>  s alpha(W)sub(W)2
          568577 ALPHA
             299 ALPHAS
          568687 ALPHA
                 (ALPHA OR ALPHAS)
           53200 SUB
              40 SUBS
           53237 SUB
                 (SUB OR SUBS)
         2785091 2
L6              1 ALPHA(W)SUB(W)2

=> s alpha(W)sup(W)2
          568577 ALPHA
             299 ALPHAS
          568687 ALPHA
                 (ALPHA OR ALPHAS)
             946 SUP
              18 SUPS
             960 SUP
                 (SUP OR SUPS)
         2785091 2
L7              0 ALPHA(W)SUP(W)2

=> s alpha(W)2
          568577 ALPHA
             299 ALPHAS
          568687 ALPHA
                 (ALPHA OR ALPHAS)
         2785091 2
L8        37612 ALPHA(W)2

=> s alpha2
L9         6800 ALPHA2

=> s l6 or l7 or l8 or l9
L10       40883 L6 OR L7 OR L8 OR L9

=> s l5 and l10
L11       12003 L5 AND L10

=> s integrin
          18159 INTEGRIN
           7861 INTEGRINS
L12       21022 INTEGRIN
                (INTEGRIN OR INTEGRINS)

=> s l11 and l12
L13         395 L11 AND L12

=> s glomerulopath10
10 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=>  s glomerulopath?
L14        1963 GLOMERULOPATH?

=> s glomerulonephropath?
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L15         174 GLOMERULONEPHROPATH?

=> s nephropath?
L16       24060 NEPHROPATH?

=> s renopath?
L17          10 RENOPATH?

=> s glomerulonephrit?
L18       15752 GLOMERULONEPHRIT?

=> s nephrit?
L19       20666 NEPHRIT?

=> s l14 or l15 or l16 or l17 or l18 or l19
L20       53371 L14 OR L15 OR L16 OR L17 OR L18 OR L19

=> s l13 and l20
L21          10 L13 AND L20

=> save temp l21
ENTER NAME OR (END):alpha/a
ANSWER SET L21 HAS BEEN SAVED AS 'ALPHA/A'

=> d l21 1-10 dn ti au so ab

L21  ANSWER 1 OF 10  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN   PREV200200491816
TI   Pattern of renal beta1(alpha1 - alpha6) integrins
     distribution in IgA nephropathy and Henoch-Schoenlein
     nephritis.
AU   Wagrowska-Danilewicz, Malgorzata; Danilewicz, Marian (1)
SO   Polish Journal of Pathology, (2002) Vol. 53, No. 2, pp. 51-57. print.
     ISSN: 1233-9687.
AB   Immunoperoxidase staining was carried out using monoclonal antibodies
     against integrins alpha1beta1, alpha2beta1, alpha3beta1,
     alpha4beta1, alpha5beta1 and alpha6beta1 on renal biopsy specimens from
     patients with IgA nephropathy (IgAN, n=15) and Henoch-Schoenlein
     nephritis (HSN, n=10). The basic pattern of glomerular and
     tubulointerstitial (alpha1 - beta6)beta1 integrin
     distribution was similar in both studied groups, however increase in
     mesangial alpha1beta1 integrin immunoexpression in biopsies from
     HSN patients as compared to biopsies from IgAN patients was observed.
     There were no statistical differences in the intensity of
     tubulointerstitial (alpha1 - alpha6)beta1 integrins
     immunolabelling in renal tissue between IgAN and HSN patients. The similar
     pattern of distribution of beta1 integrins in renal tissue in
     IgAN and HSN patients may support the hypothesis of common pathogenesis of
     IgAN and HSN. Upregulation of alpha1beta1 integrin on mesangial
     regions in biopsy specimens in patients with HSN may be connected to the
     much more florid glomerular changes in renal tissue in this type of
     glomerulonephritis than in IgAN.

L21  ANSWER 2 OF 10  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN   PREV200200075327
TI   Localization of extracellular matrix receptors in ICGN mice, a strain of
     mice with hereditary nephrotic syndrome.
AU   Uchio-Yamada, Kozue; Manabe, Noboru (1); Yamaguchi, Misuzu; Akashi,
     Naotsugu; Goto, Yasufumi; Yamamoto, Yoshie; Ogura, Atsuo; Miyamoto, Hajime
SO   Journal of Veterinary Medical Science, (November, 2001) Vol. 63, No. 11,
     pp. 1171-1178. print.
     ISSN: 0916-7250.
AB   Fibrotic degeneration was examined in the kidneys of ICR-derived
     glomerulonephritis (ICGN) mice, a novel inbred mouse line with a
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hereditary nephrotic syndrome of unknown etiology considered to be a good model of human idiopathic nephrotic syndrome. In the present study, we histochemically revealed changes in accumulation of extracellular matrix (ECM) components and in localization of **integrins**, cellular receptors for ECM, in the kidneys of ICGN mice with the progression of renal failure. Excessive accumulation of basement membrane (laminin and collagen IV) and interstitial (type III collagen) ECM components were demonstrated in the glomeruli and tubulointerstitum of ICGN mice. Marked deposition of type I collagen and tenascin was seen only in the glomeruli of ICGN mice but not in those of ICR mice as normal controls. Increased expression of **integrin alpha1**-, **alpha2**-, alpha5- and beta1-subunits in glomeruli with fibrotic degeneration and abnormal distribution of alpha6-subunit were noted in the kidneys of ICGN mice. Excessive laminin, a ligand of alpha6beta1-**integrin**, was demonstrated on the tubular basement membrane, but alpha6-subunit diffusely disappeared on the basal side of the tubular epithelial cells. We presumed that abnormal **integrin** expression in renal tubules causes epithelial cell detachment, and consequently tubular **nephropathy**, and results in disorder of ECM metabolism causing excessive accumulation of ECM components in the kidneys of ICGN mice.

L21 ANSWER 3 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN PREV200000430928
TI Distinct structural forms of type I collagen modulate cell cycle regulatory proteins in mesangial cells.
AU Schoecklmann, Harald O. (1); Lang, Stefan; Kralewski, Martina; Hartner, Andrea; Luedke, Andrea; Sterzel, R. Bernd
SO Kidney International, (September, 2000) Vol. 58, No. 3, pp. 1108-1120. print.
ISSN: 0085-2538.
AB Background: Extracellular matrix molecules profoundly regulate cell behavior, including proliferation. In **glomerulonephritis**, type I collagen accumulates in the mesangium and is constantly structurally modified and degraded during the course of the disease. Methods: We studied how two structurally distinct forms of type I collagen, monomer versus polymerized fibrils, affect cell proliferation, mitogen-activated protein kinase (MAPK) activation, and expression of G1-phase regulatory proteins in cultured rat mesangial cells (MCs). To analyze the possible involvement of collagen-binding **integrins** in type I collagen-derived growth signals further, distribution patterns of **integrin** chains were examined by immunocytochemistry. Results: Polymerized type I collagen completely prevented the increase of DNA synthesis and cell replication induced by 5% fetal calf serum (FCS) or 25 ng/mL platelet-derived growth factor (PDGF) in MCs on monomer type I collagen. Protein expression of cyclins D1 and E was markedly down-regulated in MCs plated on polymerized type I collagen for eight hours in 5% FCS, as compared with MCs on monomer type I collagen. Incubation with 5% FCS reduced expression of the cdk-inhibitor protein p27Kip1 on monomer but not on polymerized type I collagen. Moreover, polymerized type I collagen markedly reduced cyclin E-associated kinase activity in the presence of 5% FCS. Polymerized type I collagen diminished the PDGF-induced phosphorylation and nuclear translocation of p42/p44 MAPK, but did not affect phosphorylation of PDGF beta-receptors. In MCs plated on monomer type I collagen, **alpha1**, **alpha2**, and beta1 **integrin** chains were recruited into focal contacts. However, on polymerized type I collagen, **alpha2** and beta1, but not **alpha1**, **integrin** chains were condensed into focal contacts. Conclusions: The growth-inhibitory effect of polymerized type I collagen is characterized by rapid changes of expression and/or activation of MAPK and G1-phase regulators and could result from the lack of alpha1beta1 **integrin** signaling in MCs on polymerized type I collagen. Conceivably, deposition of polymerized type I collagen might reflect a reparative response to control MC replication in glomerular inflammation.

L21 ANSWER 4 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN PREV199799652434
TI Importance of the tubulointerstitium in human **glomerulonephritis**. II. Distribution of **integrin** chains beta-1, **alpha-1** to 6 and alpha-V.
AU Roy-Chaudhury, Prabir; Hillis, Graham; McDonald, Stuart; Simpson, John G.; Power, David A. (1)
SO Kidney International, (1997) Vol. 52, No. 1, pp. 103-110. ISSN: 0085-2538.
AB Accumulation of extracellular matrix is important in the progression of **glomerulonephritis**. Since adherent cell types utilize **integrins** to bind and organize extracellular matrix proteins, we have assessed expression of the **alpha-1** **integrins** in sequential sections from 85 human renal biopsies and 4 normal kidneys by immunohistochemical staining. Our results demonstrate strong correlations between expression of the alpha-5 chain within the interstitium, the alpha-V chain on proximal and distal tubular epithelium and the presence of chronic histological damage. Moreover, staining for interstitial alpha-5 and proximal and distal tubular alpha-V were also strongly associated with expression of certain adhesion molecules (ICAM-1, VCAM-1, E-selectin and L-selectin) and the presence of macrophages within the interstitium, which have been linked, in an earlier study, with the degree of chronic histological damage and disease progression. However, in contrast to our earlier study of adhesion molecules, there were also associations between expression of **integrin** chains within the glomerulus and tubulointerstitium. For example, there were strong positive associations between staining for alpha-5 on glomerular endothelium and its expression on extraglomerular vascular endothelium and between both mesangial **alpha-1** and podocyte alpha-3 and tubular staining for the common **alpha-1** subunit. While the functional significance of these associations is obscure, they suggest some kind of communication between cells in different sites in the kidney. There were also positive associations between staining for different **integrins** within the glomerulus, notably mesangial cell staining for **alpha-2**, glomerular endothelial cell staining for alpha-5 and glomerular epithelial cell alpha-3. These results suggest that there is a coordinated upregulation of **integrin** expression both within the tubulointerstitium and the glomerulus and that at least some of these **integrins** (interstitial alpha-5 and distal tubular alpha-V) are associated with the expression of other adhesion molecules, macrophage infiltration and the presence of markers of disease progression (interstitial fibrosis and tubular atrophy).

L21 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN PREV199799611731
TI Expression of beta-1 **integrins** in IgA **nephropathy**.
AU Hillis, G. S. (1); Roy-Chaudhury, P.; Duthie, L. A.; Stewart, K. N.; Brown, P. A. J.; Simpson, J. G.; MacLeod, A. M.
SO Nephrology Dialysis Transplantation, (1997) Vol. 12, No. 6, pp. 1137-1142. ISSN: 0931-0509.
AB Aim. To compare the expression of beta-1 **integrins** in renal biopsies from patients with IgA **nephropathy** with that found in normal human kidney. Methods. Thirty renal biopsies from patients with IgA disease plus six control specimens were stained with monoclonal antibodies directed against the **alpha-1**, **alpha-2**, alpha-3, alpha-4, alpha-5, alpha-6, alpha-v, and beta-1 **integrin** chains using the alkaline phosphatase anti-alkaline phosphatase (APAAP) technique. The intensity of **integrin** expression was graded semiquantitatively by a pathologist unaware of the antibody used. Results. Glomerular crescents stained strongly for alpha-3, alpha-v, and beta-1, but **integrin** expression was greatly reduced or absent in fibrotic glomeruli. There were no alterations in the intensity of mesangial cell staining for any of the **integrins**

tested. There was accentuated staining for the **alpha-2**, alpha-5, alpha-v, and beta-1 chains in areas of interstitial scarring plus **alpha-2**, alpha-3, alpha-v, and beta-1 on damaged tubules. Inflammatory cells expressed alpha-4, alpha-5, and beta-1. Conclusions. In IgA **nephropathy** the interstitium is the main site of altered beta-1 **integrin** expression. Glomerular crescents also express several beta-1 **integrins**, but we found no differences in the intensity of **integrin** expression on mesangial cells. Altered beta-1 **integrin** expression may play a role in tubulointerstitial scarring in IgA disease. Thus modulation of **integrin** expression might attenuate this process.

L21 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN PREV199799372830
TI Distribution of **integrin** subunits in human diabetic kidneys.
AU Jin, Dong Kyu; Fish, Alfred J.; Wayner, Elizabeth A.; Mauer, Michael; Setty, Suman; Tsilibary, Effie; Kim, Youngki (1)
SO Journal of the American Society of Nephrology, (1996) Vol. 7, No. 12, pp. 2636-2645.
ISSN: 1046-6673.
AB **Integrins** are cell-surface protein receptors that participate In cell adhesion to multiple extracellular matrix ligands, and consist of alpha and beta chain heterodimers. This study examined altered **integrin** distribution in diabetic **nephropathy** by investigating 12 human diabetic kidney biopsies, which were compared with normal human kidney. Diabetic **nephropathy** is characterized by mesangial expansion and progressive thickening of the glomerular basement membrane. Based on morphometric studies of mesangial expansion, diabetic **nephropathy** was determined to be moderate or severe. Three different patterns (P) of altered intensity of **integrin** staining were observed. In the mesongial **integrin** P, the intensity of **integrin** subunit staining of mesangial cells (**alpha-1**, **alpha-2**, alpha-3, beta-1, alpha-v, alpha-v-beta-5) was increased in moderate diabetic **nephropathy** and further increased in severe diabetic **nephropathy**. In the epithelial **integrin** P, **integrin** subunits localized to epithelial cells (alpha-v, beta-3, alpha-v-beta-3, alpha-v-beta-5) were increased to the same extent in moderate and severe diabetic **nephropathy**. In the endothelial **integrin** P, **integrin** subunits localized to endothelial cells (alpha-3, alpha-5, alpha-6, beta-1) were increased in moderate diabetic **nephropathy** but returned to normal kidney staining intensity in severe diabetic **nephropathy**. From these observations, it was concluded that there is significant alteration in the expression of **integrin** subunits in diabetic **nephropathy** that is related to the severity of diabetic mesangial expansion. Additionally, the spectrum of **integrin** subunit alteration appears to be unique to individual glomerular cell types. Given the role of **integrins** in cell-surface interactions with extracellular matrix components, abnormalities in the expression of these molecules may be important in the pathogenesis of diabetic **nephropathy**.

L21 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN PREV199799310952
TI Renal expression of **integrin** genes is altered in insulin dependent diabetes (IDDM.
AU Setty, S. (1); Wang, H.; Mauer, M.; Tsilibary, E. C.
SO Journal of the American Society of Nephrology, (1996) Vol. 7, No. 9, pp. 1878.
Meeting Info.: 29th Annual Meeting of the American Society of Nephrology New Orleans, Louisiana, USA November 3-6, 1996
ISSN: 1046-6673.

L21 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

DN PREV199699208135
TI Adhesion molecules in human crescentic **glomerulonephritis**.
AU Patey, N.; Lesavre, P.; Halbwachs-Mecarelli, L.; Noel, L. H.
SO Journal of Pathology, (1996) Vol. 179, No. 4, pp. 414-420.
ISSN: 0022-3417.
AB The expression of the intercellular adhesion molecule-1 (ICAM-1) and its ligand lymphocyte function associated antigen-1 (LFA-1 or alpha-L), the vascular cell adhesion molecule-1 (VCAM-1), endothelial leukocyte adhesion molecule-1 (ELAM-1), and the cellular receptors for extracellular matrix, **alpha-1**, **alpha-2**, alpha-3, alpha-5, alpha-6, alpha-V, beta-1, and beta-3 **integrin** subunits, was studied in 28 patients with crescentic **glomerulonephritis** (GN) related to several mechanisms: four patients with anti-glomerular basement membrane antibodies or anti-GBM disease; 16 with immune complex mediated GN; and eight with pauci-immune GN, associated with vasculitis in four cases. A three-step immunoperoxidase technique was used on sections obtained from frozen renal biopsies. At the initial stage of evolution of the lesions, all the cells of the crescents expressed the beta-1, beta-3, **alpha-1**, alpha-3, and alpha-L subunits of **integrins**, ICAM-1, and VCAM-1, and some cells expressed the **alpha-2**, alpha-5, alpha-6, and alpha-L subunits of **integrins** along the plasma membrane. At a later stage, when the crescents were fibrocellular, alpha-3 and alpha-1 subunit expression was polarized, localized mainly in front of the extracellular matrix. In fibrotic crescents, the **alpha-2**, alpha-5, alpha-6, and alpha-L chains were no longer detected, and VCAM-1 and ICAM-1 expression was decreased. VCAM-1 and ELAM-1 appeared on endothelial cells of peritubular capillaries in relation to the appearance of infiltrating inflammatory cells. The results of this study show that several adhesion molecules were expressed on cells forming crescents and were modified during crescent evolution; that these molecules were up-regulated on endothelial cells in relation to the severity of the inflammatory response; and that whatever the mechanism of the **glomerulonephritis**, adhesion molecule expression was identical. It can be postulated that adhesion molecules play a role in crescentic **glomerulonephritis**. Better knowledge of these molecules in human **glomerulonephritis** may open the way to a new therapeutic approach.

L21 ANSWER 9 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN PREV199598461852
TI Beta-1 and beta-3 **integrin** upregulation in rapidly progressive **glomerulonephritis**.
AU Baraldi, A. (1); Zambruno, G.; Furci, L.; Ballestri, M.; Tombesi, A.; Ottani, D.; Lucchi, L.; Lusvarghi, E.
SO Nephrology Dialysis Transplantation, (1995) Vol. 10, No. 7, pp. 1155-1161.
ISSN: 0931-0509.
AB The expression and distribution pattern of beta-1(**alpha**-**1**-alpha-6) and alpha-v-beta-3 **integrins** and ICAM-1 and VCAM-1 counter receptors were evaluated by an immunohistochemical technique on eight renal samples from patients affected by rapidly progressive **glomerulonephritis** (RPGN) of different aetiologies. In all cases **integrins** and counterreceptors displayed similar patterns. On tubular cells of renal cortex, a marked upregulation of **alpha-2**-beta-1, alpha-3-beta-1, alpha-55-beta-1, alpha-v-beta-3 **integrins** and VCAM-1 was observed with as many as 60-90% of tubular cross-sections labelled, while a strong ICAM-1 reactivity was limited to the luminal surface. The same adhesion molecules were also uniformly expressed on crescentic cells. In glomeruli, **integrin** upregulation occurred only on apparently preserved capillary tufts, i.e. in an early stage of lesion, while collapsed and sclerotic tufts showed a reduced **integrin** expression. In addition a morphometric study of extracellular matrix (EM) proteins cellular fibronectin and tenascin showed a 9.56 +- 1.9-fold and 3.35 +- 0.6-fold increase respectively in these proteins, as compared to normal

kidney (P lt 0.001). The upregulation of alpha-v-beta-3 on podocytes might play a role in the adhesion of crescentic cells. An increased production of cytokines, in particular transforming growth factors, might induce an-merited deposition of EM proteins and upregulation of beta-1 and beta-3, **integrins** in RPGN.

L21 ANSWER 10 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DN PREV199497247929
TI Extracellular matrix accumulation in immune-mediated tubulointerstitial injury.
AU Tang, Winson W.; Feng, Lili; Xia, Yiyang; Wilson, Curtis B. (1)
SO Kidney International, (1994) Vol. 45, No. 4, pp. 1077-1084.
ISSN: 0085-2538.
AB The accumulation of excessive extracellular matrix (ECM) following tubular injury likely represents an imbalance between ECM production and degradation. We assessed the temporal relationship between the accumulation of ECM, cell adhesion molecules, matrix degrading proteinases, and their inhibitors in a rat model of anti-tubular basement membrane (TBM) antibody-associated tubulointerstitial **nephritis** (TIN) by the RNase protection assay and immunohistochemistry. There was an increase in the steady state expression of fibronectin (FN) and **alpha-2**(IV) collagen mRNAs beginning on day 7 with the onset of neutrophil infiltration. An increase in **alpha-1**(III) collagen and **alpha-1-integrin** did not occur until days 9 and 10, respectively, at which time mononuclear leukocytes were the predominant infiltrating cell. Increased levels of FN, **alpha-1**(III), **alpha-2**(IV) and **alpha-1-integrin** mRNAs occurred through day 14. By immunohistochemistry, increased accumulation of collagen IV, heparan sulfate proteoglycan, and laminin were detected along the thickened TBM; collagens I and III were immunolocalized within the tubulointerstitium, while FN was present in both the TBM and interstitium in rats with TIN on day 14. The increase in matrix accumulation was associated with little or no increase in proteinases. u-PA transcripts fell beginning on day 8, with recovery to control values by day 12. Transin mRNA was found at low levels only on days 8 and 9, and the protein could not be detected by Western blotting. In contrast, these changes were associated with an increase in proteinase inhibitors, so that TIMP and PAI-1 mRNAs increased beginning on day 7 and persisted through day 14. PAI-1 mRNA correlated with biologic activity, while TIMP was immunolocalized within the peritubular endothelium and infiltrating leukocytes. These data demonstrate a temporal association between ECM accumulation, a minimal change in proteinase, and an increase in proteinase inhibitors.

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	35.38	35.59

STN INTERNATIONAL LOGOFF AT 10:20:55 ON 10 DEC 2002